

Can we assess the risk of embolic complications of cardioversion?

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Atrial fibrillation (AF) is the most common sustained arrhythmia. It occurs in up to 2% of the general population, particularly in elderly patients, and remains the major challenge in cardiology.¹ AF is associated with increased risk of thromboembolic events, frequently leading to large strokes.¹⁻³ Decision making for thromboprophylaxis needs to balance the risk of stroke against the risk of major bleeding.² Stroke risk is a continuum but current guidelines recommend focusing on the identification of “truly low-risk” patients with AF, in whom anticoagulation could be safely avoided.^{2,4,5}

The CHA₂DS₂-VASc score is related to long-term risk of ischemic stroke in patients with nonvalvular AF and not receiving anticoagulation. The score is based on simple clinical parameters, not taking into account anatomic data available from cardiac imaging studies. Nevertheless, the CHA₂DS₂-VASc score has been well validated and became the most common tool used for the assessment of long-term stroke risk in clinical practice.⁴

AF predisposes to blood stasis and may lead to atrial thrombus formation.³ In up to 90% of cases, it is located in the left atrial appendage (LAA) and can be mobilized when the sinus rhythm is restored. However, there is no validated scoring system assessing the risk of thromboembolic complications of cardioversion. The recommended way to avoid stroke is not to perform cardioversion if AF persists for more than 48 hours and if there had been no previous anticoagulant treatment lasting at least 3 to 4 weeks.^{1,6} The number of such patients is substantial, and in clinical practice, they are either discharged home with an anticoagulant prescribed or require transesophageal echocardiography (TEE) to exclude LAA thrombus (LAAT). It is clear that such an approach causes delays and generates costs and logistic problems, and a delay in cardioversion may decrease the number of successful cardioversion procedures. This is why, Jaroch

et al⁴ attempted to identify additional predictors allowing to evaluate embolic risk in this setting.

Transthoracic echocardiography (TTE) has a very low sensitivity in identifying LAAT but provides information on cardiac structure and function, which helps assess the embolic risk.³⁻⁸ Nevertheless, TTE-derived parameters (ie, ejection fraction [EF] <40%) play only a minor role in the current risk classification scheme. The presented study identifies left ventricular (LV) and left atrial size (LV end-diastolic dimension [LVEDd] >52 mm; left atrial diameter >51mm) as independent predictors of the LAA thrombogenic milieu. The calculated cut-off value for the LVEDd is puzzling, as 52 mm is within the normal range.⁹ The chamber size and volume indexation to the body surface area may better predict LAAT, but this was not analyzed in the study group.¹⁰ A reduced EF (being also a part of the CHA₂DS₂-VASc score) was associated with LAAT in a univariate but not in a multivariate analysis. It should be mentioned, however, that the Teicholz's formula used in the study is currently not recommended, and a more precise Simpson's method should be used instead.^{5,8} The authors did not analyze the LV mass index or LV diastolic function, although some studies suggested a discriminative capability of such parameters in the prediction of LAAT.¹¹

TEE is a very useful tool for excluding LAAT before cardioversion.³ In the Stroke Prevention in Atrial Fibrillation (SPAF) substudy, the presence of LAAT, LAA peak flow velocity of less than 27 cm/s, and aortic plaque were all independently associated with thromboembolic events.¹² In the current era, when left atrial occluders have become available, more focus has been placed on LAA imaging, and important limitations of LAA evaluation on TEE were documented compared with computed tomography, magnetic resonance imaging, and contrast angiography. In less experienced hands, TEE may provide both false-positive

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(caused by reverberations) and false-negative results (related to a complex LAA anatomy). TEE can detect LAAT, which precludes cardioversion, but on the other hand, if LAAT is not visualized on TEE, it does not mean that thromboembolic complications of cardioversion may be completely excluded. The LAA morphology may be very complex, with several curved, trabeculated, and poorly visible lobes. Another reason for embolic complications that cannot be predicted by TEE is the prolonged LAA stunning, which occurs after cardioversion and causes delayed clot formation.

It should be noted that Jaroch et al⁴ described the correct TEE method for LAA scanning. They obtained not only the 2 perpendicular LAA views used to position the imaging plane well so as to avoid reverberations, but also performed continuous 0° to 180°-plane sweep to look at the LAA structure from all possible dimensions.¹³ They could not use 3-dimensional echocardiography, which may be very helpful in difficult cases. Of note, they had to exclude a number of patients in whom 2 experts did not reach consensus about the presence of LAAT. There are several other TEE factors not analyzed in the present study, for example, the anatomical type of the LAA. Patients with “chicken-wing” LAA morphology are less likely to have an embolic event compared with those with 2 other types of morphology. Moreover, emptying velocities reflect the mechanical function of the LAA, and low LAA velocities (<20 cm/s) correlate with the presence of spontaneous echo contrast and thrombus formation.¹⁴ Currently, several new methods for the evaluation of LAA function (LAA EF, wall deformation) are under evaluation.

The CHA₂DS₂-VASc score calculated in the study group did not discriminate the presence of LAAT. Even in patients with a CHA₂DS₂-VASc score of 0, spontaneous echo contrast was found. Among patients with a CHA₂DS₂-VASc score of 1, almost 19% had an LAAT identified. Importantly, this scale was developed to assess the risk of stroke (not LAAT), in patients not receiving anticoagulation. In the study group, more than half of the patients received some (probably not adequate) anticoagulation. It may have influenced LAAT.^{4,5} Recent studies have shown low-to-moderate usefulness of the CHADS₂ and CHA₂DS₂-VASc scores to predict the presence of LAAT and failed to identify precisely the very low-risk group.¹⁵

The number of cardioversion candidates is large and it is not reasonable to perform a semi-invasive TEE study in all of them. Not only the number of TEE probes but also the number of echocardiographers experienced in the precise evaluation of the LAA is limited, and false results may lead to errors.^{1,2} The Cardioversion Safety Score (CATES) has been recently proposed to detect patients with very low risk for thromboembolism who can be spared TEE.¹⁶ The score includes C-reactive protein levels, indexed atrial volume, troponin, AF duration, and history of embolism. It was evaluated in a relatively small group of 180 patients.¹⁶

The presented study found that a long history of AF (>1 year) and aortic calcification (but not the history of vascular disease) were also predictors of LAAT.

The presented work contributes to the ongoing discussion about the risk stratification and optimal management of patients with AF. It confirms the fact that the CHA₂DS₂-VASc score is not suited for the prediction of LAAT before cardioversion. New methods combining clinical and echocardiographic findings would be very useful in this clinical scenario. The study suggests that the enlarged left ventricle and left atrium as well as a long history of AF and the presence of aortic calcifications may be considered risk factors for the formation of LAAT. The CATES score proposes an alternative method for precardioversion assessment. However, all these findings still need further validation in large population samples to confirm its capability of selecting a very low-risk group of patients that can be spared TEE. In the meantime, the existing AF strategies and guidelines remain valid.

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